%^Dialog;HighlightOn=*;HighlightOff=*;

Connecting via Winsock to Dialog

Logging in to Dialog

Trying 31060000009998...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
****
ENTER PASSWORD:
****
Welcome to DIALOG

Dialog level 04.20.00D

Last logoff: 03jan05 16:17:09
Logon file405 04jan05 09:47:42
*** ANNOUNCEMENT ***
***
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
***
NEW FILES RELEASED
***German Patents Fulltext (File 324)

***Beilstein Abstracts (File 393)
***Beilstein Facts (File 390)
***Beilstein Reactions (File 391)
***
***
REMOVED
***Info Sci & Tech Abs (File 202)
***Internet & Personal Comp Abs (File 233)
***CanCorp Financials (File 491)

>>> Enter BEGIN HOMEBASE for Dialog Announcements <<< >>> of new databases, price changes, etc. <<< ****
HILIGHT set on as '*'
KWIC is set to 50.
* * *
SYSTEM:HOME
Cost is in DialUnits
Menu System II: D2 version 1.7.9 term=ASCII
*** DIALOG HOMEBASE(SM) Main Menu ***
Information:
1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.) 5. Product Descriptions
Connections:
6. DIALOG(R) Document Delivery
7. Data Star(R)

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/H = Help /L = Logoff /NOMENU = Command Mode

Enter an option number to view information or to connect to an online service. Enter a BEGIN command plus a file number to search a database (e.g., B1 for ERIC).
? b 410

04jan05 09:47:42 User217743 Session D651.1
$0.00 0.206 DialUnits FileHomeBase
$0.00 Estimated cost FileHomeBase
$0.00 Estimated cost this search
$0.00 Estimated total session cost 0.206 DialUnits
File 410:Chronolog(R) 1981-2004/Nov
(c) 2004 The Dialog Corporation

Set Items Description
--- ----- -----------
? set hi *;set hi *
HILIGHT set on as '*'*
HILIGHT set on as ''
? b155
04jan05 09:47:47 User217743 Session D651.2
$0.00 0.100 DialUnits File410
$0.00 Estimated cost File410
$0.02 TELNET
$0.02 Estimated cost this search
$0.02 Estimated total session cost 0.306 DialUnits
File 155:MEDLINE(R) 1951-2004/Dec W1
(c) format only 2004 The Dialog Corp.
*File 155: Medline has stopped updating as of December 7, 2004. Please see HELP NEWS 155 for details.

Set Items Description
--- ----- -----------
? recall temp
Name Date Time Size
------- -------- -------- ----
TD074 03jan05 16:17:09 14
? exs TD074
HILIGHT set on as '%'
KWIC is set to 50.
431 CTGF
395124 FAMILY
S1 76 CTGF AND FAMILY
76 S1
65067 CYTOKINE
S2 1 S1 AND CYTOKINE
65067 CYTOKINE
395124 FAMILY

```
    S3   3813  CYTOKINE AND FAMILY
         3813  S3
          431  CTGF
    S4      1  S3 AND CTGF
         3813  S3
         5021  NOV
    S5      1  S3 AND NOV
         3813  S3
            1  SEF10
          148  CYR61
    S6      1  S3 AND (SEF10 OR CYR61)
    S7      1  SEF10
          380  SEF
      1249930  10
    S8      1  SEF()10
    S9      0  CYR?61
          148  CYR61
           45  CYR
        95181  61
            3  CYR(W)61
   S10    150  CYR61 OR CYR()61
          150  S10
       395124  FAMILY
   S11     81  S10 AND FAMILY
           81  S11
         5021  NOV
   S12     49  S11 AND NOV
? t s12/3,ab/1-5

 12/3,AB/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

17298807   PMID: 15579080
  Structural Basis and Therapeutic Implication of the Interaction of CCN Proteins with Glycoconjugates.
  Desnoyers Luc
  Departments of Molecular Oncology and Pathology, Genentech Inc., m/s 42, 1 DNA Way, South San Francisco, California 94080, USA. desnoyer@gene.com.
Current pharmaceutical design (Netherlands)  2004,  10 (31)  p3913-28, ISSN 1381-6128   Journal Code: 9602487
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: In Data Review
  The CCN %family% of growth factors is composed of six structurally related proteins including the cysteine-rich 61 (%Cyr61%), connective tissue growth factor (CTGF), nephroblastoma overexpressed (%NOV%), Wnt-1 induced secreted protein-1 (WISP-1), WISP-2 and WISP-3. Each %family% member consists of four conserved cysteine rich modular domains with sequence similarity to the insulin like growth factor binding proteins, von Willebrand factor, thrombospondin repeat and the growth factors cysteine knot. The CCN proteins demonstrate a wide variety of biological activities regulating cell adhesion, proliferation, survival, migration, invasion in vitro and tumorigenesis and angiogenesis in vivo. Both cancer promoting and inhibiting roles were proposed for several CCN proteins suggesting that contextual factors could regulate their activities. Consistent with this hypothesis, structural and experimental evidence indicate that the function of these proteins is modulated by their interaction with sulfated glycosaminoglycans. Because the CCN proteins are implicated in the tumorigenic process, they are potential targets for the development of cancer therapeutics. Modulation of their glycosaminoglycan interaction by exogenous, highly sulfated polysaccharides, oligosaccharides or glycosaminoglycan mimetics could prevent their participation in cancer progression. Understanding the structural requirements for their polysaccharide interaction should provide important information to generate glycosaminoglycan-based cancer therapeutics targeting the CCN %family% of proteins.

 12/3,AB/2
DIALOG(R)File 155:MEDLINE(R)
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17143116   PMID: 15308622
  Identification of a novel integrin alphavbeta3 binding site in CCN1 ( %CYR61% ) critical for pro-angiogenic activities in vascular endothelial cells.
  Chen Ningyu; Leu Shr-Jeng; Todorovic Viktor; Lam Stephen C-T; Lau Lester F
  Department of Biochemistry and Molecular Genetics, University of Illinois College of Medicine, Chicago 60607-7170, USA.
  Journal of biological chemistry (United States)  Oct 15 2004,  279 (42)  p44166-76, ISSN 0021-9258   Journal Code: 2985121R
  Contract/Grant No.: CA46565; CA; NCI; CA80080; CA; NCI; HL41793; HL; NHLBI
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: In Process
  CCN1 (%CYR61% ) is a matricellular inducer of angiogenesis essential for successful vascular development. Though devoid of the canonical RGD sequence motif recognized by some integrins, CCN1 binds to, and functions through integrin alphavbeta3 to promote pro-angiogenic activities in activated endothelial cells. In this study we identify a 20-residue sequence, V2 (NCKHQCTCIDGAVGCIPLCP), in domain II of CCN1 as a novel binding site for integrin alphavbeta3. Immobilized synthetic V2 peptide supports alphavbeta3-mediated cell adhesion; soluble V2
```

peptide inhibits endothelial cell adhesion to CCN1 and the homologous %family% members CCN2 (connective tissue growth factor, CTGF) or CCN3 (%NOV%) but not to collagen. These activities are obliterated by mutation of the aspartate residue in the V2 peptide to alanine. The corresponding D125A mutation in the context of the N-terminal half of CCN1 (domains I and II) greatly diminished direct solid phase binding to purified integrin alphavbeta3 and abolished alphavbeta3-mediated cell adhesion activity. Likewise, soluble full-length CCN1 with the D125A mutation is defective in binding purified alphavbeta3 and impaired in alphavbeta3-mediated pro-angiogenic activities in vascular endothelial cells, including stimulation of cell migration and enhancement of DNA synthesis. In contrast, immobilized full-length CCN1-D125A mutant binds alphavbeta3 and supports alphavbeta3-mediated cell adhesion similar to wild type CCN1. These results indicate that V2 is the primary alphavbeta3 binding site in soluble CCN1, whereas additional cryptic alphavbeta3 binding site(s) in the C-terminal half of CCN1 becomes exposed when the protein is immobilized. Together, these results identify a novel and functionally important binding site for integrin alphavbeta3 and provide a new approach for dissecting alphavbeta3-specific CCN1 functions both in cultured cells and in the organism.

12/3,AB/3
DIALOG(R)File 155:MEDLINE(R)

17029209 PMID: 15213231
CCN3 (%NOV%) interacts with connexin43 in C6 glioma cells: possible mechanism of connexin-mediated growth suppression.
Fu Christine T; Bechberger John F; Ozog Mark A; Perbal Bernard; Naus Christian C
Department of Anatomy and Cell Biology, University of British Columbia, Vancouver V6T 1Z3, British Columbia, Canada.
Journal of biological chemistry (United States) Aug 27 2004, 279 (35) p36943-50, ISSN 0021-9258 Journal Code: 2985121R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Many tumor cells exhibit aberrant gap junctional intercellular communication, which can be restored by transfection with connexin genes. We have previously discovered that overexpression of connexin43 (Cx43) in C6 glioma cells not only reduces proliferation but also leads to production of soluble growth-inhibitory factors. We identified that several members of the CCN (%Cyr61%/connective tissue growth factor/nephroblastoma-overexpress ed) %family% are up-regulated following Cx43 expression, including CCN3 (%NOV%). We now report evidence for an association between CCN3 and Cx43. Western blot analysis demonstrated that the 48-kDa full-length CCN3 protein was present in the lysate and conditioned medium of growth-suppressed C6-Cx43 cells, as well as primary astrocytes, but not in C6 parental and human glioma cells. Immunocytochemical examination of CCN3 revealed diffuse localization in parental C6 cells, whereas transfection of C6 cells with Cx43 (C6-Cx43) or with a modified Cx43 tagged to green fluorescent protein on its C terminus (Cx43-GFP) resulted in punctate staining, suggesting that CCN3 co-localizes with Cx43 in plaques at the plasma membrane. In cells expressing a C-terminal truncation of Cx43 (Cx43Delta244-382), this co-localization was lost. Glutathione S-transferase pull-down assay and co-immunoprecipitation demonstrated that CCN3 was able to physically interact with Cx43. In contrast, CCN3 was not found to associate with Cx43Delta244-382. Similar experiments revealed that CCN3 did not co-localize or associate with other connexins, including Cx40 or Cx32. Taken together, these data support an interaction of CCN3 with the C terminus of Cx43, which could play an important role in mediating growth control induced by specific gap junction proteins.

12/3,AB/4
DIALOG(R)File 155:MEDLINE(R)

17006069 PMID: 15331410
WISP-1 is an osteoblastic regulator expressed during skeletal development and fracture repair.
French Dorothy M; Kaul Raji J; D'Souza Aloma L; Crowley Craig W; Bao Min; Frantz Gretchen D; Filvaroff Ellen H; Desnoyers Luc
Department of Pathology, Genentech Incorporated, South San Francisco, California, USA.
American journal of pathology (United States) Sep 2004, 165 (3) p855-67, ISSN 0002-9440 Journal Code: 0370502
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Wnt-1-induced secreted protein 1 (WISP-1) is a member of the CCN (connective tissue growth factor, %Cyr61%, %NOV%) %family% of growth factors. Experimental evidence suggests that CCN %family% members are involved in skeletogenesis and bone healing. To investigate the role of WISP-1 in osteogenic processes, we characterized its tissue and cellular expression and evaluated its activity in osteoblastic and

chondrocytic cell culture models. During embryonic development, WISP-1 expression was restricted to osteoblasts and to osteoblastic progenitor cells of the perichondral mesenchyme. In vitro, we showed that WISP-1 expression in differentiating osteoblasts promotes BMP-2-induced osteoblastic differentiation. Using in situ and cell binding analysis, we demonstrated WISP-1 interaction with perichondral mesenchyme and undifferentiated chondrocytes. We evaluated the effect of WISP-1 on chondrocytes by generating stably transfected mouse chondrocytic cell lines. In these cells, WISP-1 increased proliferation and saturation density but repressed chondrocytic differentiation. Because of the similarity between skeletogenesis and bone healing, we also analyzed WISP-1 spatiotemporal expression in a fracture repair model. We found that WISP-1 expression recapitulates the pattern observed during skeletal development. Our data demonstrate that WISP-1 is an osteogenic potentiating factor promoting mesenchymal cell proliferation and osteoblastic differentiation while repressing chondrocytic differentiation. Therefore, we propose that WISP-1 plays an important regulatory role during bone development and fracture repair.

12/3,AB/5
DIALOG(R)File 155:MEDLINE(R)
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16202077 PMID: 15053922
Three members of the connective tissue growth factor %family% CCN are differentially regulated by mechanical stress.
Schild Christof; Trueb Beat
ITI Research Institute, University of Bern, P.O. Box 54, Murteustr. 35, CH-3010 Bern, Switzerland.
Biochimica et biophysica acta (Netherlands) Apr 1 2004, 1691 (1) p33-40, ISSN 0006-3002 Journal Code: 0217513
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Expression of connective tissue growth factor (CTGF), a member of the CCN gene %family% , is known to be significantly induced by mechanical stress. We have therefore investigated whether other members of the CCN gene %family%, including %Cyr61% and %Nov% , might reveal a similar stress-dependent regulation. Fibroblasts growing under stressed conditions within a three-dimensional collagen gel showed at least a 15 times higher level of %Cyr61% mRNA than cells growing under relaxed conditions. Upon relaxation, the decline of the %Cyr61% mRNA to a lower level occurred within 2 h, and was thus quicker than the response of CTGF. The regulation was fully reversible when stress was reapplied. Thus, %Cyr61% represents another typical example of a stress-responsive gene. The level of the %Nov% mRNA was low in the stressed state, but increased in the relaxed state. This CCN gene therefore shows an inverted regulation relative to that of %Cyr61% and CTGF. Inhibition of protein kinases by means of staurosporine suppressed the stress-induced expression of %Cyr61% and CTGF. Elevated levels of cAMP induced by forskolin mimicked the effects of relaxation on the regulation of %Cyr61%, CTGF and %Nov%. Thus, adenylate cyclase as well as one or several protein kinases might be involved in the mechanoregulation of these CCN genes.
? s ccn and family
228 CCN
395124 FAMILY
S13 99 CCN AND FAMILY
? s ccn()family
228 CCN
395124 FAMILY
S14 63 CCN()FAMILY
? s s14 and py<1999
63 S14
11859639 PY<1999
S15 3 S14 AND PY<1999
? t s15/3,ab/all

15/3,AB/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

14042900 PMID: 9742130
Identification of rCop-1, a new member of the CCN protein family, as a negative regulator for cell transformation.
Zhang R; Averboukh L; Zhu W; Zhang H; Jo H; Dempsey P J; Coffey R J; Pardee A B; Liang P
Vanderbilt Cancer Center, Department of Cell Biology, Vanderbilt University, Nashville, Tennessee 37232, USA.
Molecular and cellular biology (UNITED STATES) Oct %1998%, 18 (10) p6131-41, ISSN 0270-7306 Journal Code: 8109087
Contract/Grant No.: CA68485; CA; NCI; CA74067; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
By using a model system for cell transformation mediated by the cooperation of the activated H-ras oncogene and the inactivated p53 tumor suppressor gene, rCop-1 was identified by mRNA differential display as a gene whose expression became lost after cell transformation. Homology analysis indicates that rCop-1

belongs to an emerging cysteine-rich growth regulator family called CCN, which includes connective-tissue growth factor, CYR61, CEF10 (v-src inducible), and the product of the nov proto-oncogene. Unlike the other members of the CCN gene family, rCop-1 is not an immediate-early gene, it lacks the conserved C-terminal domain which was shown to confer both growth-stimulating and heparin-binding activities, and its expression is lost in cells transformed by a variety of mechanisms. Ectopic expression of rCop-1 by retroviral gene transfers led to cell death in a transformation-specific manner. These results suggest that rCop-1 represents a new class of %CCN% %family% proteins that have functions opposing those of the previously identified members.

15/3,AB/2
DIALOG(R)File 155:MEDLINE(R)

13762929 PMID: 9449709
Expression of the Elm1 gene, a novel gene of the CCN (connective tissue growth factor, Cyr61/Cef10, and neuroblastoma overexpressed gene) family, suppresses In vivo tumor growth and metastasis of K-1735 murine melanoma cells.
Hashimoto Y; Shindo-Okada N; Tani M; Nagamachi Y; Takeuchi K; Shiroishi T ; Toma H; Yokota J
Biology Division, National Cancer Center Research Institute, 1-1, Tsukiji 5-chome, Chuo-ku, Tokyo 104, Japan.
Journal of experimental medicine (UNITED STATES) Feb 2 %1998%, 187 (3) p289-96, ISSN 0022-1007
Journal Code: 2985109R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
We previously isolated a partial cDNA fragment of a novel gene, Elm1 (expressed in low-metastatic cells), that is expressed in low-metastatic but not in high-metastatic K-1735 mouse melanoma cells. Here we determined the full-length cDNA structure of Elm1 and investigated the effect of Elm1 expression on growth and metastatic potential of K-1735 cells. The Elm1 gene encodes a predicted protein of 367 amino acids showing approximately 40% amino acid identity with the CCN (connective tissue growth factor [CTGF], Cyr61/Cef10, neuroblastoma overexpressed gene [Nov]) family proteins, which consist of secreted cysteine-rich proteins with growth regulatory functions. Elm1 is also a cysteine-rich protein and contains a signal peptide and four domains conserved in the %CCN% %family% proteins. Elm1 was highly conserved, expressed ubiquitously in diverse organs, and mapped to mouse chromosome 15. High-metastatic K-1735 M-2 cells, which did not express Elm1, were transfected with an Elm1 expression vector, and several stable clones with Elm1 expression were established. The in vivo growth rates of cells expressing a high level of Elm1 were remarkably slower than those of cells expressing a low level of Elm1. Metastatic potential of transfectants was reduced in proportion to the level of Elm1 expression. Thus, Elm1 is a novel gene of %CCN% %family% that can suppress the in vivo growth and metastatic potential of K-1735 mouse melanoma cells.

15/3,AB/3
DIALOG(R)File 155:MEDLINE(R)

13306823 PMID: 8975721
Genomic structure and chromosomal mapping of the mouse nov gene.
Snaith M R; Natarajan D; Taylor L B; Choi C P; Martinerie C; Perbal B; Schofield P N; Boulter C A
Department of Genetics, University of Cambridge, United Kingdom.
Genomics (UNITED STATES) Dec 15 %1996%, 38 (3) p425-8, ISSN 0888-7543
Journal Code: 8800135
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The nov gene encodes a cysteine-rich protein that is overexpressed in avian nephroblastomas. It is a member of the %CCN% %family% of proteins, all of which are involved in cell growth. Genomic and cDNA clones encompassing the mouse nov gene have been isolated and characterized. The mouse nov gene is highly conserved with the human and chick nov genes at the level of nucleotide sequence and genomic organization. The exon structure reflects the modular organization of the NOV protein in a number of structural domains. These are highly conserved with other members of the %CCN% %family% , as is the distribution of 38 of its 40 cysteine residues. The nov gene maps to chromosome 15, between D15 Mit 153 and D15 Mit 183, in a region of conserved synteny with human chromosome 8.
? s ccn/ti and family/ti not s15
41 CCN/TI
68249 FAMILY/TI
3 S15
S16 20 CCN/TI AND FAMILY/TI NOT S15
? t s16/3,ab/all

16/3,AB/1
DIALOG(R)File 155:MEDLINE(R)

16202077 PMID: 15053922

Three members of the connective tissue growth factor %family% %CCN% are differentially regulated by mechanical stress.
Schild Christof; Trueb Beat
ITI Research Institute, University of Bern, P.O. Box 54, Murteustr. 35, CH-3010 Bern, Switzerland.
Biochimica et biophysica acta (Netherlands) Apr 1 2004, 1691 (1) p33-40, ISSN 0006-3002 Journal Code: 0217513
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Expression of connective tissue growth factor (CTGF), a member of the CCN gene family, is known to be significantly induced by mechanical stress. We have therefore investigated whether other members of the CCN gene family, including Cyr61 and Nov, might reveal a similar stress-dependent regulation. Fibroblasts growing under stressed conditions within a three-dimensional collagen gel showed at least a 15 times higher level of Cyr61 mRNA than cells growing under relaxed conditions. Upon relaxation, the decline of the Cyr61 mRNA to a lower level occurred within 2 h, and was thus quicker than the response of CTGF. The regulation was fully reversible when stress was reapplied. Thus, Cyr61 represents another typical example of a stress-responsive gene. The level of the Nov mRNA was low in the stressed state, but increased in the relaxed state. This CCN gene therefore shows an inverted regulation relative to that of Cyr61 and CTGF. Inhibition of protein kinases by means of staurosporine suppressed the stress-induced expression of Cyr61 and CTGF. Elevated levels of cAMP induced by forskolin mimicked the effects of relaxation on the regulation of Cyr61, CTGF and Nov. Thus, adenylate cyclase as well as one or several protein kinases might be involved in the mechanoregulation of these CCN genes.

16/3,AB/2
DIALOG(R)File 155:MEDLINE(R)

15822629 PMID: 15018819
The expression of chicken NOV, a member of the %CCN% gene %family%, in early stage development.
Katsube K; Chuai M L; Liu Y C; Kabasawa Y; Takagi M; Perbal B; Sakamoto K Molecular Pathology, Graduate School of Tokyo Medical and Dental University, Yushima, Bunkyo-ku, Tokyo 113-8549, Japan.
Gene expression patterns - GEP (Netherlands) Aug 2001, 1 (1) p61-5, ISSN 1567-133X Journal Code: 101167473
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Data Review
The nephroblastoma overexpressed gene, NOV, is a member of the CCN gene family. We investigated the NOV gene expression pattern in the chicken during early stage embryogenesis. Several embryonic structures showed a distinct expression pattern. The initial expression was detected in Hensen's node (Hamburger and Hamilton stage (HH) 5). The expression was noted in the presumptive notochord and floor plate forming cells. The expression on the left side was more elongated posteriorly, a type of left-right asymmetry. Chicken NOV gene expression in the forming notochord and floor plate was observed until HH 18. The expression was also detected in the ventral area of the mesencephalon and isthmus at HH 14-16.

16/3,AB/3
DIALOG(R)File 155:MEDLINE(R)

15726799 PMID: 14648665
NOV (CCN3) regulation in the growth plate and %CCN% %family% member expression in cartilage neoplasia.
Yu Chunying; Le Anh-Thy; Yeger Herman; Perbal Bernard; Alman Benjamin A Program in Developmental Biology, The Hospital for Sick Children, 555 University Avenue, Toronto, Ontario, M5G 1X8, Canada.
Journal of pathology (England) Dec 2003, 201 (4) p609-15, ISSN 0022-3417 Journal Code: 0204634
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Growth plate chondrocytes undergo a coordinated differentiation process resulting in terminal differentiation and new bone formation. Enchondromas are pre-malignant, benign cartilaginous lesions that arise from growth plate chondrocytes that fail to undergo terminal differentiation. NOV (nephroblastoma overexpressed) is a member of the CCN family of proteins, which share a common multi-modular organization. While the role of NOV in chondrocyte development and cartilage neoplasia is not known, other CCN family members play a role in chondrocyte differentiation, or are differentially regulated in cartilage neoplasia. In embryonic murine growth plates, NOV was expressed in pre-hypertrophic and early hypertrophic chondrocytes. PTHrP treatment (which inhibits terminal differentiation) decreased NOV expression in murine femurs maintained in organ culture, and decreased the activity of a NOV reporter construct in vitro. Expression of the CCN family members NOV, CTGF, CYR61, and WISP-1 was examined in 15 chondrosarcomas of various grades and in three enchondromas. Expression of all of the family members

was lower in the higher-grade tumours. As identification of the grade of cartilage neoplasia can sometimes be difficult using histology alone, the level of expression of CCN family members could be a useful adjunct in the determination of tumour grade. Copyright 2003 John Wiley & Sons, Ltd.

16/3,AB/4
DIALOG(R)File 155:MEDLINE(R)
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14474008 PMID: 10471507
Mutations in the %CCN% gene %family% member WISP3 cause progressive pseudorheumatoid dysplasia.
Hurvitz J R; Suwairi W M; Van Hul W; El-Shanti H; Superti-Furga A; Roudier J; Holderbaum D; Pauli R M; Herd J K; Van Hul E V; Rezai-Delui H; Legius E; Le Merrer M; Al-Alami J; Bahabri S A; Warman M L Department of Genetics and Center for Human Genetics, Case Western Reserve University School of Medicine and University Hospitals of Cleveland, Cleveland, Ohio 44106, USA.
Nature genetics (UNITED STATES) Sep 1999, 23 (1) p94-8, ISSN 1061-4036 Journal Code: 9216904
Contract/Grant No.: AR43827; AR; NIAMS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Members of the CCN (for CTGF, cyr61/cef10, nov) gene family encode cysteine-rich secreted proteins with roles in cell growth and differentiation. Cell-specific and tissue-specific differences in the expression and function of different CCN family members suggest they have non-redundant roles. Using a positional-candidate approach, we found that mutations in the CCN family member WISP3 are associated with the autosomal recessive skeletal disorder progressive pseudorheumatoid dysplasia (PPD; MIM 208230). PPD is an autosomal recessive disorder that may be initially misdiagnosed as juvenile rheumatoid arthritis. Its population incidence has been estimated at 1 per million in the United Kingdom, but it is likely to be higher in the Middle East and Gulf States. Affected individuals are asymptomatic in early childhood. Signs and symptoms of disease typically develop between three and eight years of age. Clinically and radiographically, patients experience continued cartilage loss and destructive bone changes as they age, in several instances necessitating joint replacement surgery by the third decade of life. Extraskeletal manifestations have not been reported in PPD. Cartilage appears to be the primary affected tissue, and in one patient, a biopsy of the iliac crest revealed abnormal nests of chondrocytes and loss of normal cell columnar organization in growth zones. We have identified nine different WISP3 mutations in unrelated, affected individuals, indicating that the gene is essential for normal post-natal skeletal growth and cartilage homeostasis.

16/3,AB/5
DIALOG(R)File 155:MEDLINE(R)
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14299810 PMID: 10204117
The connective tissue growth factor/cysteine-rich 61/nephroblastoma overexpressed (%CCN%) %family%.
Brigstock D R
Department of Surgery, Ohio State University, Columbus 43210, USA.
brigstod@pediatrics.ohio-state.edu
Endocrine reviews (UNITED STATES) Apr 1999, 20 (2) p189-206, ISSN 0163-769X Journal Code: 8006258
Contract/Grant No.: HD-30334; HD; NICHD
Document type: Journal Article; Review; Review, Academic Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

16/3,AB/6
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

14277346 PMID: 10094812
The %CCN% %family% of angiogenic regulators: the integrin connection. Lau L F; Lam S C
Department of Molecular Genetics, Department of Pharmacology, University of Illinois at Chicago College of Medicine, Chicago, Illinois, 60607-7170, USA.
LFLau@uic.edu
Experimental cell research (UNITED STATES) Apr 10 1999, 248 (1) p44-57, ISSN 0014-4827 Journal Code: 0373226
Contract/Grant No.: CA46565; CA; NCI; CA80080; CA; NCI; HL41793; HL; NHLBI
Document type: Journal Article; Review; Review, Academic Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

16/3,AB/7
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

12471475 PMID: 12904165
The %CCN% %family%: a new stimulus package.
Brigstock D R
Department of Surgery, The Ohio State University,

Columbus, Ohio 43212, USA.
brigstocd@pediatrics.ohio-state.edu
Journal of endocrinology (England) Aug 2003, 178 (2) p169-75, ISSN 0022-0795 Journal Code: 0375363
Contract/Grant No.: AA12817-02; AA; NIAAA
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The CCN family comprises cysteine-rich 61 (CYR61/CCN1), connective tIssue growth factor (CTGF/CCN2), nephroblastoma overexpressed (NOV/CCN3), and Wnt-induced secreted proteins-1 (WISP-1/CCN4), -2 (WISP-2/CCN5) and -3 (WISP-3/CCN6). These proteins stimulate mitosis, adhesion, apoptosis, extracellular matrix production, growth arrest and migration of multiple cell types. Many of these activities probably occur through the ability of CCN proteins to bind and activate cell surface integrins. Accumulating evidence supports a role for these factors in endocrine pathways and endocrine-related processes. To illustrate the broad role played by the CCN family in basic and clinical endocrinology, this Article highlights the relationship between CCN proteins and hormone action, skeletal growth, placental angiogenesis, IGF-binding proteins and diabetes-induced fibrosis.

16/3,AB/8
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12418864 PMID: 12695522
CCN3 (NOV) is a novel angiogenic regulator of the %CCN% protein %family%.
Lin Cristiane G; Leu Shr-Jeng; Chen Ningyu; Tebeau Christopher M; Lin Shao-Xia; Yeung Cho-Yau; Lau Lester F
Department of Molecular Genetics, University of Illinois at Chicago College of Medicine, Chicago, Illinois, 60607, USA.
Journal of biological chemistry (United States) Jun 27 2003, 278 (26) p24200-8, ISSN 0021-9258 Journal Code: 2985121R
Contract/Grant No.: CA46565; CA; NCI; CA78044; CA; NCI; CA80080; CA; NCI; CA91376; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
CCN3 (NOV) is a matricellular protein of the CCN family, which also includes CCN1 (CYR61), CCN2 (CTGF), CCN4 (WISP-1), CCN5 (WISP-2), and CCN6 (WISP-3). During development, CCN3 is expressed widely in derivatives of all three germ layers, and high levels of expression are observed in smooth muscle cells of the arterial vessel wall. Altered expression of CCN3 has been observed in a variety of tumors, including hepatocellular carcinomas, Wilm's tumors, Ewing's sarcomas, gliomas, rhabdomyosarcomas, and adrenocortical carcinomas. To understand its biological functions, we have investigated the activities of purified recombinant CCN3. We show that in endothelial cells, CCN3 supports cell adhesion, induces directed cell migration (chemotaxis), and promotes cell survival. Mechanistically, CCN3 supports human umbilical vein endothelial cell adhesion through multiple cell surface receptors, including integrins alphavbeta3, alpha5beta1, alpha6beta1, and heparan sulfate proteoglycans. In contrast, CCN3-induced cell migration is dependent on integrins alphavbeta3 and alpha5beta1, whereas alpha6beta1 does not play a role in this process. Although CCN3 does not contain a RGD sequence, it binds directly to immobilized integrins alphavbeta3 and alpha5beta1, with half-maximal binding occurring at 10 nm and 50 nm CCN3, respectively. Furthermore, CCN3 induces neovascularization when implanted in rat cornea, demonstrating that it is a novel angiogenic inducer. Together, these findings show that CCN3 is a ligand of integrins alphavbeta3 and alpha5beta1, acts directly upon endothelial cells to stimulate pro-angiogenic activities, and induces angiogenesis in vivo.

16/3,AB/9
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12347401 PMID: 12717393
Human WISP1v, a member of the %CCN% %family%, is associated with invasive cholangiocarcinoma.
Tanaka Shinji; Sugimachi Keishi; Kameyama Toshifumi; Maehara Shin-Ichiro; Shirabe Ken; Shimada Mitsuo; Wands Jack R; Maehara Yoshihiko Department of Surgery and Science, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan. shinjit@surg2.med.kyushu-u.ac.jp Hepatology (Baltimore, Md.) (United States) May 2003, 37 (5) p1122-9 , ISSN 0270-9139 Journal Code: 8302946
Contract/Grant No.: CA-37511; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Family members of the connective tissue growth factor, cysteine-rich 61, nephroblastoma over-expressed gene (CCN) encode cysteine-rich secreted proteins with roles in human fibrotic disorders and tumor progression. In this study, we identified a CCN family member, WISP1v, as over-expressed in human cholangiocarcinomas. Genetic analysis of WISP1v was performed on surgically resected specimens of cholangiocarcinoma. The WISP1v biological effects were analyzed using the HuCCT1 human cholangiocarcinoma cell

line. The WISP1v gene was expressed in 19 of 39 cholangiocarcinoma tissues (49%) but not in normal livers. Expression of WISP1v was significantly associated with lymphatic and perineural invasion of tumor cells (P <.05), as well as a poor clinical prognosis (P <.01). In the intraductal papillary cholangiocarcinomas, WISP1v was detected only in the cases with duct wall invasion but not in the cases without duct wall invasion (P <.05). No mutation of WISP1v gene was detected in the examined samples. In vitro analysis revealed that WISP1v stimulated the invasive phenotype of cholangiocarcinoma cells with activation of both p38 and p42/p44 mitogen-activated protein kinases (MAPKs). Furthermore, WISP1v-induced cholangiocarcinoma invasion was significantly suppressed by the p38 MAPK inhibitor SB203580 but not by the p42/p44 MAPK kinase (MEK) inhibitor PD98059. Our findings suggest that WISP1v-mediated signaling is involved in the generation of invasive cellular properties and leads to progression of cholangiocarcinoma.

16/3,AB/10
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12307720 PMID: 12665625
Report on the second international workshop on the %CCN% %family% of genes.
Perbal B; Brigstock D R; Lau L F
Laboratoire d'Oncologie Virale et Moleculaire, UFR de Biochimie, Universite Paris 7-D Diderot, 2 Place Jussieu, 75005 Paris, France. perbal@ccr.jussieu.fr
Molecular pathology - MP (England) Apr 2003, 56 (2) p80-5, ISSN 1366-8714 Journal Code: 9706282
Document type: Congresses
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
For the second time, researchers from leading laboratories in the CCN field gathered in Saint-Malo, France, to participate in the Second International Workshop on the CCN family of genes. In addition to the regular research communications, meeting highlights included the inauguration of the first CCN newsletter (http://ccnnewsletter.free.fr) and the recognition of the International CCN Society (http://www.ccnsociety.jussieu.fr) as an important medium for the exchange of scientific knowledge and resources in the CCN field. Once more, the high quality of scientific communications and individual interactions set the stage for an extremely fruitful meeting.

16/3,AB/11
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12065710 PMID: 12390824
Progress in the study of %CCN% %family%.
Su Bing-Yin; Cai Wen-Qin
Department of Histology and Embryology, Third Military Medical University, Chongqing 400038, China. subingyin@yahoo.com.cn Di yi jun yi da xue xue bao = Academic journal of the First Medical College of PLA (China) Feb 2002, 22 (2) p179-83, ISSN 1000-2588 Journal Code: 9426110
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The last 5-6 years have seen the emergence of a new gene family that currently comprises CTGF, Cyr61, nov, elm1, HICP and WISP-3. The translational products of most CCN family members are secreted proteins that contain 343 to 381 amino-acid residues to compose 4 distinct structural modules, each having 38 conserved cysteine residues. These proteins have a variety of properties to affect the cellular behaviors such as growth, differentiation, adhesion and locomotion. They may play important roles in pregnancy, development and differentiation, angiogenesis, wound repair, fibrotic disorders, inflammation and tumor genesis.

16/3,AB/12
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11870614 PMID: 12064632
Determination of a potential role of the %CCN% %family% of growth regulators in connexin43 transfected C6 glioma cells.
McLeod T L; Bechberger J F; Naus C C
Department of Anatomy and Cell Biology, The University of Western Ontario, London, Canada.
Cell communication & adhesion (Switzerland) 2001, 8 (4-6) p441-5, ISSN 1541-9061 Journal Code: 101096596
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Tumour cells often exhibit erratic cell growth, as well as decreased gap junctional intercellular communication (GJIC). C6 glioma cells are characterized by low levels of gap junction mRNA and protein, and decreased amounts of GJIC when compared with astrocytes. Previous work has shown that C6 glioma cells transfected with connexin43 (C6-Cx43) exhibit

decreased proliferation in vivo and in vitro, as well as genes that are differentially expressed between these cells. In this study, RNA levels of two CCN (connective tissue growth factor [CTGF], Cyr61/Cef-10, nephroblastoma overexpressed [NOV]) gene family members are shown to be upregulated in C6-Cx43 cells: Cyr61 and Nov. Cyr61 has previously been shown to increase adhesion, migration and growth in many cell types, whereas NOV has growth suppressive capacities. Cyr61 RNA expression is shown here to respond to serum in quiescent cells in an immediate early gene fashion, independent of Cx43 expression. In contrast, Nov RNA levels remain constant, reflective of transfected Cx43 expression. Furthermore, confocal microscopy indicates that NOV colocalizes with Cx43 plaques at the cell membrane. These findings provide insight into the possible role of Nov and Cyr61 in tumour cells.

16/3,AB/13
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11572382 PMID: 11598125
Cyr61, a member of %CCN% %family%, is a tumor suppressor in non-small cell lung cancer.
Tong X; Xie D; O'Kelly J; Miller C W; Muller-Tidow C; Koeffler H P Division of Hematology/Oncology, Cedars-Sinai Medical Center, UCLA School of Medicine, Davis Bldg. Rm. 5022, 8700 Beverly Blvd., Los Angeles, CA 90048, USA. xiangjuntong@hotmail.com
Journal of biological chemistry (United States) Dec 14 2001, 276 (50) p47709-14, ISSN 0021-9258 Journal Code: 2985121R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Cysteine-rich protein 61 (Cyr61) is a member of a family of growth factor-inducible immediate-early genes. It regulates cell adhesion, migration, proliferation, and differentiation and is involved in tumor growth. In our experiments, the role of Cyr61 in non-small cell lung cancer (NSCLC) was examined. Expression of Cyr61 mRNA was decreased markedly in four of five human lung tumor samples compared with their normal matched lung samples. NSCLC cell lines NCI-H520 and H460, which have no endogenous Cyr61, formed 60-90% fewer colonies after being transfected with a Cyr61 cDNA expression vector than cells transfected with the same amount of empty vector. After stable transfection of a Cyr61 cDNA expression vector, proliferation of both H520-Cyr61 and H460-Cyr61 sublines decreased remarkably compared with the cells stably transfected with empty vector. The addition of antibody against Cyr61 partially rescued the growth suppression of both H520-Cyr61 and H460-Cyr61 cells. Cell cycle analysis revealed that both H520-Cyr61 and H460-Cyr61 cells developed G(1) arrest, prominently up-regulated expression of p53 and p21(WAF1), and had decreased activity of cyclin-dependent kinase 2. The increase of pocket protein pRB2/p130 was also detected in these cells. Notably, both of the Cyr61-stably transfected lung cancer cell lines developed smaller tumors than those formed by the wild-type cells in nude mice. Taken together, we conclude that Cyr61 may play a role as a tumor suppressor in NSCLC.

16/3,AB/14
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11469277 PMID: 11577174
The Twisted gastrulation %family% of proteins, together with the IGFBP and %CCN% families, comprise the TIC superfamily of cysteine rich secreted factors.
Vilmos P; Gaudenz K; Hegedus Z; Marsh J L
Department of Developmental and Cell Biology, University of California Irvine, Irvine, CA 92697, USA.
Molecular pathology - MP (England) Oct 2001, 54 (5) p317-23, ISSN 1366-8714 Journal Code: 9706282
Contract/Grant No.: HD36049; HD; NICHD; HD36081; HD; NICHD Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
AIMS: To analyse the similarities between the Twisted gastrulation (TSG) proteins known to date; in addition, to determine phylogenetic relations among the TSG proteins, and between the TSGs and other protein families--the CCN (for example, CCN2 (CTGF), CCN1 (CYR61), and CCN3 (NOV)) and IGFBP (insulin-like growth factor binding protein) families. METHODS: TBLASTN and FASTA3 were used to identify new tsg genes and relatives of the TSG family. The sequences were aligned with ClustalW. The predictions of sites for signal peptide cleavage, post-translational modifications, and putative protein domains were carried out with software available at various databases. Unrooted phylogenetic trees were calculated using the UPGMA method. RESULTS: Several tsg genes from vertebrates and invertebrates were compared. Alignment of protein sequences revealed a highly conserved family of TSG proteins present in both vertebrates and invertebrates, whereas the slightly less well conserved IGFBP and CCN proteins are apparently present only in vertebrates. The TSG proteins display strong homology among themselves and they are composed of a putative signal peptide at the N-terminus followed by a cysteine rich (CR) region, a conserved domain devoid

of cysteines, a variable midregion, and a C-terminal CR region. The most striking similarity between the TSGs and the IGFBP and CCN proteins occurs in the N-terminal conserved cysteine rich domain and the characteristic 5' cysteine rich domain(s), spacer region, and 3' cysteine rich domain structure. CONCLUSION: The family of highly conserved TSG proteins, together with the IGFBP and CCN families, constitute an emerging multigene superfamily of secreted cysteine rich factors. The TSG branch of the superfamily appears to pre-date the others because it is present in all species examined, whereas the CCN and IGFBP genes are found only in vertebrates.

16/3,AB/15
DIALOG(R)File 155:MEDLINE(R)

11400729 PMID: 11495815
[The %CCN% %family% of cell growth regulators: a new %family% of normal and pathologic cell growth and differentiation regulators: lessons from the first international workshop on %CCN% gene %family%]
Les proteines %CCN%: une nouvelle famille de regulateurs de la croissance et de la differenciation cellulaire normale et pathologique: les enseignements du premier congres international sur la famille des genes %CCN%.
Perbal B
Laboratoire d'oncologie virale et moleculaire, UFR de biochimie, Universite Paris 7, D. Diderot, 2, place Jussieu, 75005 Paris, France. perbal@ccr.jussieu.fr
Bulletin du cancer (France) Jul 2001, 88 (7) p645-9, ISSN 0007-4551 Journal Code: 0072416
Document type: Congresses
Languages: FRENCH
Main Citation Owner: NLM
Record type: Completed

16/3,AB/16
DIALOG(R)File 155:MEDLINE(R)

11297079 PMID: 11382921
COP-1, a member of the %CCN% %family%, is a heparin-induced growth arrest specific gene in vascular smooth muscle cells.
Delmolino L M; Stearns N A; Castellot J J
Department of Pathology, Brigham and Womens Hospital, Harvard Medical School, Boston, Massachusetts, USA.
Journal of cellular physiology (United States) Jul 2001, 188 (1) p45-55, ISSN 0021-9541 Journal Code: 0050222
Contract/Grant No.: HL49973; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Vascular smooth muscle cell (VSMC) hyperplasia is responsible for the failure of 15-30% of vascular surgical procedures such as coronary artery bypass grafts and angioplasties. We and others have shown that heparin suppresses VSMC proliferation in vivo and in cell culture. We hypothesize that heparin inhibits VSMC proliferation by binding to cell surface receptors, resulting in selective modulation of mitogenic signal transduction pathways and altered transcription of a specific subset of growth regulatory genes. To test this idea, we used subtractive hybridization to identify differentially expressed mRNAs in heparin-treated and untreated VSMC. We identified a heparin induced mRNA identical to Cop-1, a member of the CCN family of proteins which are secreted, cysteine-rich modular proteins involved in growth regulation and migration. Cop-1 from smooth muscle cells appears to have a different expression pattern and possibly different functions than Cop-1 from other cells. Cop-1 mRNA is expressed at high levels in quiescent VSMC and at low levels in proliferating VSMC, an expression pattern highly characteristic of growth arrest specific genes. Cop-1 mRNA is expressed at high levels in heparin treated VSMC and COP-1 protein is secreted into culture medium. In tissues, Cop-1 expression is observed in the uninjured rat aorta suggesting a possible role for Cop-1 in vivo. We found PDGF, but not EGF, inhibits the expression of Cop-1 in VSMC. Neither TGF-beta nor interferon-beta, two inhibitors of VSMC proliferation, were able to induce Cop-1 expression. In addition, heparin does not induce Cop-1 mRNA in endothelial cells and VSMC resistant to the antiproliferative effect of heparin. Conditioned medium from cells over-expressing COP-1 protein inhibits VSMC proliferation in culture. Together, our data indicate that COP-1 may play a role in the antiproliferative mechanism of action of heparin.

16/3,AB/17
DIALOG(R)File 155:MEDLINE(R)

11277567 PMID: 11356703
Cyr61, a member of the %CCN% %family% , is required for MCF-7 cell proliferation: regulation by 17beta-estradiol and overexpression in human breast cancer.
Sampath D; Winneker R C; Zhang Z
Women's Health Research Institute, Division of

Endocrinology, Wyeth-Ayerst Research, Inc., Radnor, Pennsylvania 19087, USA. sampatd@war.wyeth.com
Endocrinology (United States) Jun 2001, 142 (6) p2540-8, ISSN 0013-7227 Journal Code: 0375040
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Cyr61, a member of the CCN (CTGF/Cyr61/NOV) family of growth regulators, is a secreted cysteine-rich proangiogenic factor that has been implicated in tumorigenesis. Previous studies have also demonstrated that Cyr61 is regulated by 17beta-estradiol (E(2)) in the uterus. Therefore, we hypothesized that hormonal regulation of Cyr61 may be important in estrogen-dependent pathogenic processes such as breast tumorigenesis. Our study demonstrates that both Cyr61 messenger RNA and protein are induced by E(2) in MCF-7 mammary adenocarcinoma cells that primarily overexpress estrogen receptor alpha (ERalpha) in a dose-dependent and immediate early fashion. Cyr61 gene induction by E(2) is transcriptionally regulated by ERalpha as the antiestrogen, ICI 182,780, and actinomycin D blocked induction completely. In addition, Cyr61 is up-regulated in MCF-7 cells by epidermal growth factor (EGF) in an immediate early fashion as well. The functional relevance of steroid induction of Cyr61 in breast cancer cell growth is demonstrated by anti-Cyr61 neutralizing antibodies, which diminished E(2) and EGF-dependent DNA synthesis and dramatically reduced E(2)-driven cell proliferation by more than 70%. Most importantly, Cyr61 is overexpressed in 70% (28 of 40) of breast cancer patients with infiltrating ductal carcinoma and is localized exclusively to hyperplastic ductal epithelial cells. Moreover, the levels of Cyr61 protein are higher in breast tumors that are ER(+)/EGF receptor(+) than those that are ER(-)/EGF receptor(+), suggesting that estrogens may mediate Cyr61 expression in vivo. Collectively, our data suggest that Cyr61 may play a critical role in estrogen- as well as growth factor-dependent breast tumor growth.

16/3,AB/18
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11245403 PMID: 11322166
Report and abstracts on the first international workshop on the %CCN% %family% of genes.
Ayer-Lelievre C; Brigstock D; Lau L; Pennica D; Perbal B; Yeger H Molecular pathology - MP (England) Apr 2001, 54 (2) p105-20, ISSN 1366-8714 Journal Code: 9706282
Document type: Congresses; Overall
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

16/3,AB/19
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11245402 PMID: 11322165
The %CCN% %family% of genes: a brief history.
Perbal B
Molecular pathology - MP (England) Apr 2001, 54 (2) p103-4, ISSN 1366-8714 Journal Code: 9706282
Document type: Editorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

16/3,AB/20
DIALOG(R)File 155:MEDLINE(R)
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11245397 PMID: 11322167
NOV (nephroblastoma overexpressed) and the %CCN% %family% of genes: structural and functional issues.
Perbal B
Laboratoire d'Oncologie Virale et Moleculaire, UFR de Biochimie, Universite Paris 7-D, Diderot, France. bernard.perbal@wanadoo.fr Molecular pathology - MP (England) Apr 2001, 54 (2) p57-79, ISSN 1366-8714 Journal Code: 9706282
Document type: Journal Article; Review; Review, Academic Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The CCN family of genes presently consists of six distinct members encoding proteins that participate in fundamental biological processes such as cell proliferation, attachment, migration, differentiation, wound healing, angiogenesis, and several pathologies including fibrosis and tumorigenesis. Whereas CYR61 and CTGF were reported to act as positive regulators of cell growth, NOV (nephroblastoma overexpressed) provided the first example of a CCN protein with negative regulatory properties and the first example of aberrant expression being associated with tumour development. The subsequent discovery of the ELM1, rCOP1, and WISP proteins has broadened the variety of functions attributed to the CCN proteins and has extended previous observations to other biological systems. This review discusses fundamental questions regarding the regulation of CCN gene expression in normal and pathological conditions, and the structural basis for their specific biological activity. After

discussing the role of nov and other CCN proteins in the development of a variety of different tissues such as kidney, nervous system, muscle, cartilage, and bone, the altered expression of the CCN proteins in various pathologies is discussed, with an emphasis on the altered expression of nov in many different tumour types such as Wilms's tumour, renal cell carcinomas, prostate carcinomas, osteosarcomas, chondrosarcomas, adrenocortical carcinomas, and neuroblastomas. The possible use of nov as a tool for molecular medicine is also discussed. The variety of biological functions attributed to the CCN proteins has led to the proposal of a model in which physical interactions between the amino and carboxy portions of the CCN proteins modulate their biological activity and ensure a proper balance of positive and negative signals through interactions with other partners. In this model, disruption of the secondary structure of the CCN proteins induced by deletions of either terminus is expected to confer on the truncated polypeptide constitutive positive or negative activities.

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